that doubt the effectiveness of non-invasive techniques forget that it is their unfamiliarity that limits the value of myocardial perfusion imaging and not the technique itself. Instead of ignoring it they should follow the practice of their colleagues who are able to manage patients with objective measurements of myocardial perfusion rather than subjective impressions of the severity of disease made from the coronary arteriogram. They might then find that they can make many therapeutic decisions without the need to reach for their catheters, and we shall be spared the debate about the role of cardiac catheterisation in the district general hospital.

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Acromegalic heart disease

SIR,—We disagree with several of the statements in the article by Rodrigues *et al* entitled "Subclinical cardiac dysfunction in acromegaly: evidence for a specific disease of heart muscle" (1989;**62**:185–94).

In the summary Rodrigues *et al* say that "This is the first study to find evidence of subclinical cardiac diastolic dysfunction in acromegaly", and in the discussion they say that "Left ventricular diastolic function has not previously been studied in acromegaly". I would like to draw attention to the paper of Bertoni and Morandi entitled "Impaired left ventricular diastolic function in acromegaly: an echocardiographic study", where relaxation abnormalities of the left ventricle were shown in acromegalic patients in 1987.[1]

On the basis of the paper by Lie and Grossman,[2] Rodrigues *et al* suggest that the fibrosis which is partly responsible for the diastolic filling disturbance is a consequence of an inflammatory process. Van den Heuvel *et al* took biopsy specimens of the right ventricle of an acromegalic patient and later examined specimens taken at necropsy. Both specimens showed the same changes: hypertrophic myocardial fibres and some fibrous thickening of the endocardium. There was no evidence of any inflammatory changes.[3] In rats with myocardial hypertrophy caused by tumours producing growth hormone Gilbert *et al* found direct evidence that there was no underlying inflammatory process.[4]

These results prove that the main pathological background of acromegalic heart disease is left ventricular hypertrophy, with some contribution from myocardial fibrosis and the increased collagen content of the myocardium. We found the dilated form of the disease in a few of our patients but not an inflammatory process; these patients were burned-out cases with lower serum concentrations of growth hormone than those with hypertrophy.[5]

Rodrigues *et al* cited the second edition of Feigenbaum's *Echocardiography* (not published in 1979 but in 1976) as saying that echocardiography is an insensitive method of assessing left ventricular function. However, this is not the opinion given in the fourth edition published in 1986.[6] Radionuclide ventriculography is probably a better method of assessing the ejection fraction than echocardiography. But because there are several other indices of left ventricular function (for example, enlargement of the left ventricle and segmental wall movement abnormalities) echocardiography cannot be deemed to be an insensitive method of evaluating left ventricular function.[7]

In Rodrigues *et al*'s paper apart from 13 self-citations, there are only three references from 1982–84 and only one, an abstract, dated 1986. The references I cite were published between 1983 and 1987.

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This letter was shown to the authors, who reply as follows:

SIR,—In reply to Csanády's letter we would like to make the following comments. Bertoni and Morandi published a paper on left ventricular diastolic function in 1987,[1] but our study was largely completed in 1986 and was presented in part to the British Cardiac Society in December 1986.[2]

Csanády seems to disregard the seminal work on pathological findings of Lie and Grossman[3] in favour of the necropsy findings in a patient described by van den Heuvel *et al*.[4] Necropsy findings in only one patient do not disprove the findings of a large study. Furthermore, in our study we tried to assess the influence of myocardial hypertrophy by studying left ventricular mass derived echocardiographically and showed that there was no correlation between peak filling rate abnormalities and left ventricular wall thickness or left ventricular mass. Therefore, it is unlikely that the abnormal peak filling rate seen in our patients was merely a reflection of hypertrophy.

Though we agree that echocardiography can be used not only to measure ejection fraction but also to assess the anatomical consequences of left ventricular dysfunction, such as left ventricular enlargement and wall motion abnormalities, the point we made in our discussion was that subtle changes in diastolic relaxation were shown better by radionuclide ventriculography. Indeed, Feigenbaum[5] pointed out the drawbacks and pitfalls of calculating ejection fraction by cross sectional echocardiography and did not go into any detail about the assessment of subtle diastolic function by this technique. We referred to our previous validation studies in the text to establish the quantitative success and reproducible nature of the radionuclide technique for assessing subtle diastolic left ventricular dysfunction. Many methods have been used to study a large group of patients with a rare condition. Previous studies did not deal with the same questions, nor was thallium-201 imaging used to exclude obstructive coronary artery disease.[6] Thus our suggestion that radionuclide ventriculography is better than echocardiography for picking up subtle left ventricular diastolic dysfunction is valid.

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1 Bertoni PD, Morandi G. Impaired left ventricular diastolic function in acromegaly: an echocardiographic study. *Acta Cardiol (Brux)* 1987;**15**:1–10.
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Severe haemolytic anaemia after replacement of the mitral valve by a St Jude medical prosthesis

SIR,—The interesting case report by Feld and Roth (1989;**62**:475–6) of severe haemolytic anaemia after mitral valve replacement with a St Jude medical prosthesis highlights the difficulty in detecting mitral paraprosthetic leaks. Despite clinical evidence of mitral regurgitation in their patient, careful echocardiography with Doppler studies failed to detect any prosthetic abnormality and left heart catheterisation was required to show severe paraprosthetic regurgitation.

We found that transoesophageal echocardiography with a 5 MHz phased array transoesophageal transducer (HP 21362A)[1] was helpful in nine patients (mean age 58 years) with mitral prosthetic regurgitation. One of them also had severe haemolytic anaemia after the insertion of a 27 mm St Jude mitral prosthesis. Mitral regurgitation was detected by transthoracic echocardiography, includ-